Research Article

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Are herbicide-resistant crops the answer to controlling *Cuscuta*?

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Abstract

BACKGROUND: Herbicide-resistant crop technology could provide new management strategies for the control of parasitic plants. Three herbicide-resistant oilseed rape (*Brassica napus* L.) genotypes were used to examine the response of attached *Cuscuta campestris* Yuncker to glyphosate, imazamox and glufosinate. *Cuscata campestris* was allowed to establish on all oilseed rape genotypes before herbicides were applied.

RESULTS: Unattached seedlings of *C. campestris, C. subinclusa* Durand & Hilg. and *C. gronovii* Willd. were resistant to imazamox and glyphosate and sensitive to glufosinate, indicating that resistance initially discovered in *C. campestris* is universal to all *Cuscuta* species. Glufosinate applied to *C. campestris* attached to glufosinate-resistant oilseed rape had little impact on the parasite, while imazamox completely inhibited *C. campestris* growth on the imidazolinone-resistant host. The growth of *C. campestris* on glyphosate-resistant host was initially inhibited by glyphosate, but the parasite recovered and resumed growth within 3–4 weeks.

CONCLUSION: The ability of *C. campestris* to recover was related to the quality of interaction between the host and parasite and to the resistance mechanism of the host. The parasite was less likely to recover when it had low compatibility with the host, indicating that parasite-resistant crops coupled with herbicide resistance could be highly effective in controlling *Cuscuta*. Published 2009 by John Wiley & Sons, Ltd.

Keywords: *Cuscuta*; dodder; herbicide resistance; transgenic crops; amino acid inhibitors

1 INTRODUCTION

Members of the *Cuscuta* family are considered among the most damaging parasites worldwide.[1] *Cuscuta* spp. are above-ground, absolute parasites that must make contact with a susceptible host in order to survive. Once a rootless parasite seedling makes contact with a host, it develops haustoria[2,3] and connects to the host's vascular bundles,[4] forming a highly efficient absorption system that enables the parasite to divert resources from the host.[2,5] The parasite can also divert and accumulate phloem-mobile herbicides absorbed by the host. The combination of herbicide-resistant crops and phloem-mobile herbicides has been used to control parasitic plants such as *Orobanche* spp.[6] and *Striga* spp.[7]

This same combination of technologies has been less successful for control of *Cuscuta* spp. *Cuscata campestris* Yuncker was able to recover from glyphosate and sulfometuron applications when attached to herbicide-resistant sugar beet (*Beta vulgaris* L.).[8] These findings suggest that herbicide-resistant crop technology may not be as successful in managing *Cuscuta* spp. compared with its success for root parasites. This initial work also did not determine if the resistance of *C. campestris* to glyphosate and sulfometuron resulted from metabolism, lack of translocation within the parasite, sequestration or insensitive sites of action in the chloroplast. The extent to which the basic physiology of *Cuscuta* spp. differs from non-parasitic, higher plants and the relative importance of the parasite's own biosynthetic pathways have not been clearly defined. Although *Cuscuta* spp. have functional chloroplasts,[9] the plastids contain only small amounts of chlorophyll and other accessory pigments;[10–12] however, these plastids are probably more functional than previously thought with respect to protein synthesis.[13] Understanding the importance of the chloroplast in a parasite's metabolism is a significant issue, as pathways inhibited by some of the main herbicide families are located in the chloroplast (e.g. photosynthesis, amino acid, chlorophyll and lipid biosynthesis).

The three amino acid biosynthesis inhibitors examined in this study all inhibit biosynthetic pathways located in the chloroplast. Glyphosate inhibits aromatic amino acid biosynthesis by inhibiting enolpyruvylshikimate-3-phosphate synthase (EPSPS), while a large group of acetolactate synthetase (ALS) inhibitors stop the biosynthesis of branched chained amino acids. Glufosinate inhibits glutamine synthetase, the initial enzyme involved in the assimilation of inorganic nitrogen into organic forms.[14]

Although *C. campestris* has not been reported as a problem in oilseed rape, this crop was chosen as a model host because it could be easily infested with *Cuscuta* spp., but more significant is the availability of glyphosate-, imidazolinone- and glufosinate-resistant varieties. The primary objective of this research was to

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compare the efficacy of glyphosate, imazamox and glufosinate for control of *C. campestris* while attached to herbicide-resistant oilseed rape. In addition, the response of *C. campestris, C. gronovii* Willd. and *C. subinclusa* Durand & Hilg. to these herbicides as unattached seedlings was examined.

2 MATERIALS AND METHODS

2.1 Plant material

Seeds of *Cuscuta campestris* (collected in Colorado, USA), *Cuscuta gronovii* (collected in Massachusetts, USA) and *Cuscuta subinclusa* (collected in California, USA) were grown and propagated on oilseed rape in the greenhouse to maintain a homogeneous supply of viable seeds. All *Cuscuta* seeds were cleaned and acid scarified in phosphoric acid for 20 min to improve germination. Seeds of oilseed rape (cv. Hyola 420, Advanta Seeds, The Netherlands) with no herbicide resistance (WT) were used as herbicide-susceptible controls for imazamox and glufosinate treatments, and seeds of a sorghum sudangrass [*Sorghum bicolor* (L.) Moench] hybrid were used as herbicide-susceptible controls for glyphosate and imazamox. Two imidazolinone-resistant (Hylite 289 CF, Advanta Seeds, The Netherlands, and 46A76 Pioneer HB, Canada), a glyphosate-resistant (RR-DKL-35-85 Dekalb, USA) and a glufosinate-resistant (InVigor 2373, Bayer CropScience, Germany) oilseed rape were used as hosts for *C. campestris* in the herbicide efficacy assays and as herbicide-resistant references for the seedling bioassay.

2.2 Herbicides

Glyphosate-potassium 550 g AE L^{-1} SL (Roundup WeatherMAX®; Monsanto, USA), imazamox-ammonium 120 g AE L^{-1} SL (Raptor®; BASF, USA) and glufosinate-ammonium 120 g AE L^{-1} SL (Rely®; Bayer CropScience, USA) were evaluated. A non-ionic surfactant (Activator 90; Loveland Products, Inc., USA) was added at 2.5 g L^{-1} to spray solution of all herbicides; in addition, ammonium sulphate at 20 g L^{-1} was added to all glufosinate applications. In order to improve glufosinate absorption, pots were watered 1 h before treatment to increase humidity levels.[15]

2.3 Seedling bioassay

Dose–response assays were performed on the basis of the method of Tal *et al.*[16] Seeds were placed on sheets of germination paper (20 × 25 cm; Seedburo Equipment Co., USA) soaked in glyphosate (0, 0.01, 0.05, 0.1, 0.5, 1.0, 5.0, 10, 50, 100 mM), imazamox (0, 0.01, 0.1, 1.0, 10, 100, 1000, 10 000, 100 000 μM) or glufosinate (0, 0.01, 0.05, 0.1, 0.5, 1.0, 5.0, 10, 50, 100 mM) dilutions made up in 10.0 mM phosphate buffer at pH 6.0. Seeds were placed approximately 5 cm from the top of the sheet and then each sheet was rolled, secured lightly with a rubber band, placed in a glass beaker and covered with a plastic bag. Beakers were incubated in the dark at 30 °C; no additional solution was added during the experiment. *Cuscuta campestris* and *C. subinclusa* shoot lengths were measured 5 days after sowing (DAS), while *C. gronovii* shoot lengths were measured 6 DAS. The root lengths of sorghum and oilseed rape were measured 4 and 5 DAS respectively.

2.4 Pot assays

2.4.1 *Small pot assays*

Rape seeds were sown in 0.67 L plastic pots containing Metro Mix® 200 potting soil (Scott-Sierra Horticultural Products Co., USA) supplemented with 14-14-14 Osmocote® pellets (Scott-Sierra Horticultural Products Co., USA). At 5 DAS, approximately 30 acid-scarified *C. campestris* seeds were sown close to the developing oilseed rape seedlings. No *Cuscuta* seeds were sown in the control pots. Pots were kept in the greenhouse at 21–27/14–18 °C (day/night), watered as needed and fertilized with 20-20-20 Peter's fertilizer® (The Scott's Company, USA) once a week. Herbicides (commercial formulations) were applied to *Cuscuta*-infested and non-infested oilseed rape with a laboratory chain-driven sprayer calibrated to deliver 200 L ha^{-1} of glyphosate (0, 188, 315, 750, 1500 g AE ha^{-1}), imazamox (0, 10, 20, 40, 80 g AE ha^{-1}) or glufosinate (0, 50, 100, 200, 400 g AE ha^{-1}). Pots containing *C. campestris*-infested WT oilseed rape and parasite-infested imidazolinone-resistant, glyphosate-resistant and glufosinate-resistant oilseed rape were treated with imazamox, glyphosate and glufosinate respectively. Host and parasite development were monitored and graded before and after treatment. At the final assessment time, 25 days after treatment (DAT) for glufosinate, 34 DAT for imazamox and 41 DAT for glyphosate, the host plant and parasite were scored from 0 (dead) to 5 (full vigor). The host plant and *Cuscuta* tissue were harvested and separated for fresh and dry weight determinations.

2.4.2 *Large pot assays*

Rape seeds were sown in 4.8 L pots containing Metro Mix® 200 and incubated under the same conditions as described above. Three weeks after sowing, the oilseed rape was parasitized by coiling two detached *Cuscuta* shoot segments (10 cm long) around oilseed rape plants. Two weeks later, successfully parasitized plants were sprayed with imazamox (20 g AE ha^{-1}), glyphosate (750 g AE ha^{-1}) or glufosinate (400 g AE ha^{-1}). Treated pots were monitored to detect signs of recovery until 90 DAT.

2.5 Statistical analysis

Seedling dose–response assays were conducted in a completely randomized design with three replications. Using Sigma Plot 9.0 software (Systate Software Inc., USA), the shoot and root elongation data were analyzed using a sigmoidal logistic regression equation model.[17] The calculated R^2 values indicate the goodness of fit of each calculated curve from which I_{50} (the rate causing 50% reduction in tissue elongation) was predicted for each herbicide and seed variety.

Tissue fresh weight data collected for pot assays after treatment with herbicides were analyzed by ANOVA, and means were separated at the $P = 0.05$ level by Tukey's multiple comparison procedure using JMPIN 5.1 software (SAS Institute, Inc., USA).

3 RESULTS

3.1 Seedling bioassay

Cuscuta campestris, C. subinclusa and *C. gronovii* were insensitive to glyphosate and imazamox when they were grown alone without a host (Table 1). The I_{50} values of all three *Cuscuta* species were similar to those calculated for herbicide-resistant oilseed rape. The calculated I_{50} values for glyphosate were in a range similar to the I_{50} (52 mM) found for a *C. campestris* population tested in Israel.[8] The response of all *Cuscuta* species to imazamox was consistent with previous results showing that *C. campestris* seedlings can tolerate other ALS inhibitors[8]. The three *Cuscuta* species tested were extremely sensitive to glufosinate. The I_{50} values calculated for all *Cuscuta* species and WT oilseed rape were in the range

Table 1. Seedling response to amino acid biosynthesis inhibitor herbicides as determined in the germination paper assay. The I_{50} values were calculated using a log-logistic model[17] indicating the level of sensitivity to each herbicide for each variety tested (R^2 of each I_{50} calculated is indicated in parentheses)

	Herbicide I_{50}		
Genotype	Glyphosate (mM)	Imazamox (μM)	Glufosinate (μM)
C. campestris[a]	24.0 (0.96)	9185 (0.81)	0.06 (0.96)
C. gronovii[a]	13.1 (0.97)	2618 (0.93)	0.06 (0.94)
C. subinclusa[a]	21.1 (0.97)	8926 (0.87)	0.05 (0.99)
S. bicolor[b]	0.08 (0.97)	1.4 (0.97)	–
WT oilseed rape[b]	–	0.3 (0.97)	0.04 (0.91)
Imidazolinone-resistant oilseed rape[b]	–	1130 (0.92)	–
Glyphosate-resistant oilseed rape[b]	25.6 (0.89)	–	–
Glufosinate-resistant oilseed rape[b]	–	–	4.8 (0.85)

[a] Inhibition of shoot elongation.
[b] Inhibition of root elongation.

Table 2. Effectiveness of glufosinate in the control of *Cuscuta campestris* attached to a glufosinate-resistant host. Percentage of surviving *Cuscuta* plants and biomass (g fresh weight) of parasite on glufosinate-resistant oilseed rape 25 days after treatment with glufosinate (data are shown as the mean of 5–6 plants)

Glufosinate (g AE ha^{-1})	Percentage survival	Biomass (g fresh weight)[a]
0	100	9a
50	100	12a
100	100	21a
200	100	12a
400	100	7a

[a] Means followed by the same letter within a row are not different at the $P = 0.05$ level.

0.04–0.06 μM glufosinate, whereas the I_{50} of the glufosinate-resistant oilseed rape was 4.8 μM.

3.2 Pot assays

Cuscuta campestris attached to glufosinate-resistant oilseed rape was initially affected by glufosinate applications (Fig. 1B), but recovered rapidly (Fig. 1C) and by the end of the experiment had significantly reduced host biomass (data not shown). Thus, the herbicide treatment had very little impact on parasite development (Table 2). *Cuscuta campestris* attached to glufosinate-resistant oilseed rape recovered and resumed growth after treatment with 50–400 g AE ha^{-1} glufosinate (Table 2). Increasing the glufosinate rate to 800 g ha^{-1} had little effect on *C. campestris* control or reducing parasite damage to the host (data not shown). Imazamox (40 g AE ha^{-1}) inhibited *C. campestris* growth on imidazolinone-resistant oilseed rape (Fig. 2A); however, in spite of the initial damage caused to the parasite by the herbicide treatment, it was still able to reduce the biomass of the imidazolinone-resistant oilseed rape up to 80%. This host biomass reduction occurred when the parasite attachment encircled the oilseed rape stem, causing a severe restriction of stem development that lasted even after the parasite had died (Fig. 2B).

Glyphosate applied at high rates to glyphosate-resistant oilseed rape significantly reduced *C. campestris* biomass (Fig. 3A); however, at 34 DAT most of the herbicide-treated *Cuscuta* plants maintained viable and recovering apexes (Table 3; Fig. 3B).

Examination of the *C. campestris* pattern of attachment to a host over time suggests that the parasite's ability to recover from herbicide treatment is dependent on the site of the attachment. When *C. campestris* parasitizes a young developing oilseed rape plant, it penetrates either the host's lower leaf petioles or the stem. If the parasite attaches only to the lower leaves, it is likely to be shed early when these leaves undergo premature senescence. Alternatively, if the parasite coils around the stem of a young plant, it frequently kills the host by restricting stem expansion and growth. On the other hand, when *C. campestris* explants are placed on older, well-developed plants, the parasite is more likely to attach to the upper parts of the host and less likely to kill the host.

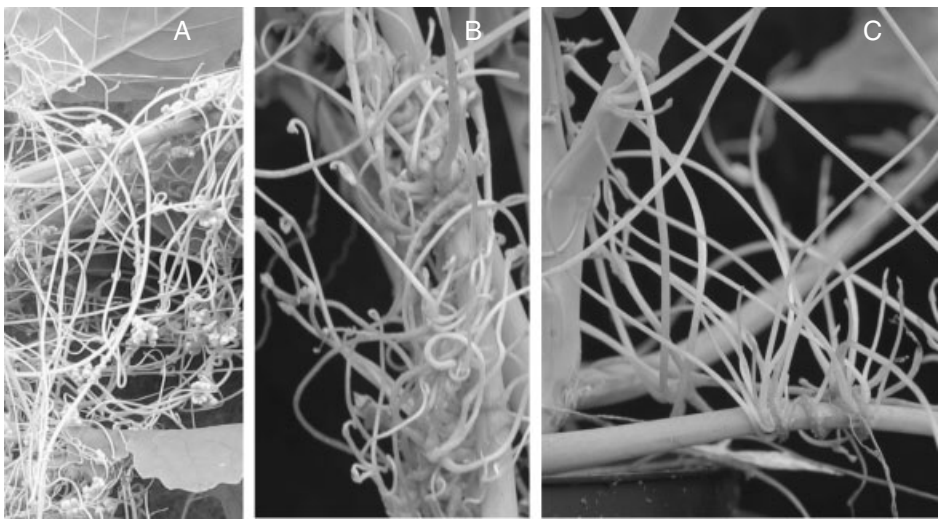

Figure 1. *Cuscuta campestris* on glufosinate-resistant oilseed rape treated with 400 g AE ha^{-1} glufosinate: **A**, control; **B**, growth inhibition; **C**, recovery of parasite.

Figure 2. *Cuscuta campestris* on imidazolinone-resistant oilseed rape: **A**, poor recovery of *C. campestris* on a host treated with 20 g AE ha^{-1} imazamox; **B**, host growth constriction by parasite.

Table 3. Effectiveness of herbicides in the control of *Cuscuta campestris* attached to herbicide-resistant hosts. Percentage of *Cuscuta* plants surviving on imidazolinone-resistant and glyphosate-resistant canola after treatment with imazamox and glyphosate 34 and 41 days after treatment respectively

Glyphosate			Imazamox	
Rate (g AE ha^{-1})	Percentage survival	Biomass (g fresh weight)[a]	Rate (g AE ha^{-1})	Percentage survival
0	100	36 a	0	100
188	80	8 b	10	0
375	100	12 b	20	0
750	60	0.1 b	40	0
1500	40	0.1 b	80	0

[a] Means followed by the same letter within a row are not different at the $P = 0.05$ level.

In order to establish whether the quality of attachment between the host and parasite determines the ability of *C. campestris* to recover from imazamox and glyphosate treatments, explants of the parasite were used to parasitize older imidazolinone-resistant and glyphosate-resistant plants. Glyphosate applied to large oilseed rape host plants infested with *C. campestris* initially inhibited the growth of the parasite for the first 3 weeks after treatment (Fig. 3A). However, it was observed that the parasite slowly recovered from the herbicide damage, resumed growth (Table 3; Fig 3B), flowered (Fig. 3C) and set seed (Fig. 3D). Only a few *C. campestris* plants were able to recover from imazamox treatment while attached to imidazolinone-resistant oilseed rape (Fig. 2B).

The fresh weight of *C. campestris* growing on both imidazolinone-resistant oilseed rape hosts was significantly lower than that of the parasite grown on all other hosts (Fig. 4). The parasite thrived on WT oilseed rape and developed well on glufosinate- and glyphosate-resistant oilseed rape, indicating a higher compatibility of the parasite for these cultivars.

4 DISCUSSION

The dose–response assays confirm that the resistance of *Cuscuta* seedlings to imazamox and glyphosate and their sensitivity to glufosinate are consistent among *C. campestris* seeds collected in Colorado and Israel,[8] and are a universal response of germinating *C. gronovii* and *C. subinclusa* seedlings tested in this study. The dose–response seedling assay has been used successfully to predict and distinguish between herbicide-resistant and herbicide-sensitive crops and weeds;[16] however, the response of *C. campestris* in the seedling assay did not coincide with the response of the parasite to the same herbicides when attached to a host. This suggests either that this assay is not sufficiently predictive to evaluate the response of *Cuscuta* to herbicides inhibiting amino acid biosynthesis or that various processes (e.g. expression of special genes) are initiated in the parasite when associated with the host plants.

In vivo shikimate and ALS assays confirm that the ALS and EPSPS enzymes are active in *C. campestris* and are sensitive to ALS inhibitors and glyphosate respectively,[8] suggesting that the tolerance of the parasite in the seedling bioassay to glyphosate and imazamox is not related to an altered target site. Ammonia accumulation has been detected by HPLC in glufosinate-treated oilseed rape and *C. campestris*, indicating that the parasite contains an active glutamine synthetase that is sensitive to glufosinate (data not shown). These data support the authors' theory that the interaction between the *Cuscuta* and host may activate different processes in the parasite.

The rapid recovery of the parasite grown on glufosinate-resistant oilseed rape treated with glufosinate, in spite of its high sensitivity to the herbicide in the seedling bioassay, is most likely a result of the mechanism of resistance in the host. Oilseed rape resistance to glufosinate was facilitated by inserting the *bar* gene, which encodes an enzyme that rapidly acetylates glufosinate to an inactive metabolite.[18] Owing to the inactivation and limited translocation of the herbicide in the host,[19] glufosinate could be prevented from being accumulated as an intact and active molecule in the parasite. Although attached *Cuscuta* plants were also sprayed with glufosinate, the amount of herbicide absorbed could not have been sufficient to kill the parasite, indicating that the herbicide primarily gets to the parasite through the host and not by direct application. Imidazolinone-resistant oilseed rape contains two modified ALS genes that encode for imidazolinone-resistant enzymes.[20] Imazamox applied to imidazolinone-resistant oilseed rape would not be readily metabolized in the host and therefore could rapidly accumulate in the attached *C. campestris*,

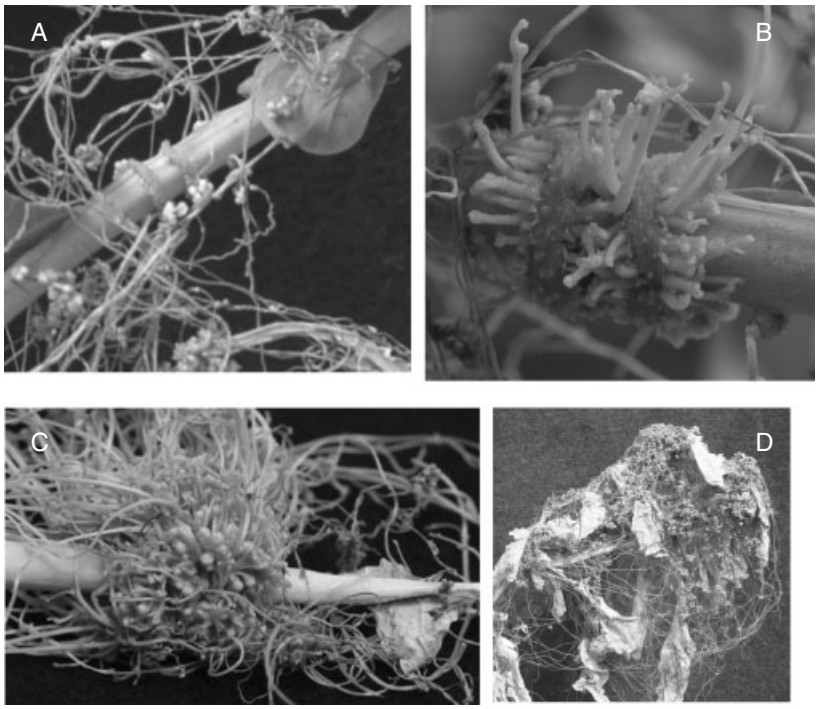

Figure 3. Response of *Cuscuta campestris* growing on a glyphosate-resistant oilseed rape host to treatment with 750g AE ha^{-1} glyphosate: **A**, 18 DAT; **B**, 29 DAT; **C**, 43 DAT; **D**, 90 DAT.

which acts as a 'supersink',[21] thus continuously exposing the parasite to a high dose of the herbicide.

Glyphosate-resistant oilseed rape contains two genetic modifications. It carries the CP4 EPSPS gene that encodes a glyphosate-resistant EPSPS, and a second gene that encodes for a glyphosate-degrading enzyme – glyphosate oxidoreductase (GOX). This gene was isolated from a common soil bacterium *Achromobacter* sp. and rapidly degrades glyphosate to non-toxic products, aminomethylphosphonic acid (AMPA) and glyoxylate.[22,23] When glyphosate is applied to glyphosate-resistant oilseed rape, part of it will be metabolized to inactive metabolites, hence reducing the amount of intact glyphosate that can be translocated to the attached *C. campestris*. Thus, the parasite would be exposed to less herbicide, resulting in reduced control and eventual recovery.

The assay comparing *C. campestris* biomass on different oilseed rape hosts indicates that, even though *C. campestris* is reported to be a non-selective parasite capable of invading many hosts,[1,24] it is not equally virulent to all potential hosts. The parasite differed in its ability to thrive on different varieties of the same host (Fig. 4). The high efficacy of imazamox on *C. campestris* (Table 3) could be due to a combination of herbicide susceptibility and reduced compatibility. Reduced compatibility might mean that a major avoidance mechanism, i.e. branch chain amino acids being provided by the host, is reduced, leading to improved herbicide efficacy. *Cuscuta campestris* appeared to be very compatible with the glyphosate-resistant oilseed rape, and this may have contributed to the parasite's ability to obtain enough nutrients from the host to recover from glyphosate treatments. Previous observations have shown that *C. campestris* was more likely to recover from herbicide treatments while attached to glyphosate-resistant sugar beet[8] than if attached to glyphosate-resistant soybean. These differences could be attributed to the quality of attachment between the host and parasite. The importance of good establishment and compatibility between the host and parasite has also been reported to enhance the translocation of labelled nitrogen from the host to *C. campestris*,[25] to increase the likelihood that *C. gronovii* would mature on a host[26] and to ensure the survival of *C. subinclusa* during hot summers.[27]

The present results suggest that the most important factor in determining the efficacy of amino acid biosynthesis inhibitors to control *Cuscuta* could be the quality of attachment between the host and parasite. The strong attachment between *C. campestris* and glufosinate-resistant oilseed rape enabled the parasite to overcome herbicide injury and to recover from high glufosinate applications. These results raise questions regarding the effectiveness of using tomato (*Solanum lycopersicum* L.) or lucerne (*Medicago sativa* L.) resistant to either ALS inhibitors or glyphosate to control *C. campestris*. Both crops are sensitive to the parasite, and it has been reported that one application of glyphosate was not sufficient to control *C. campestris* in lucerne.[24] Multiple applications of rimsulfuron were needed to suppress *Cuscuta* on tomatoes,[28] and further studies indicated that effective control of *Cuscuta* on tomato was achieved if herbicide was applied soon after attachment and before the parasite had established on the host.[29] It has also been noted that the effectiveness of *Cuscuta* control differed between fields and suggested that the differences were the result of differences among *Cuscuta* populations in the various fields.[30] As shown in the seedling assay, *C. campestris* from Israel[8] and California (Table 1) responded in the same manner to the amino acid biosynthesis inhibitors. Therefore, it is possible that the differences observed between the fields[30] actually reflected the quality of the connection between the parasite and the host. The reason *C. campestris* has a lower compatibility with imidazolinone-resistant oilseed rape is not yet known, but it is possible that the imidazolinone-resistant oil seed rape either has different levels of branched chain amino acids or carries other traits that affect the ability of the parasite to infest the host. It has been reported that slight changes in the internal salinity of *Beta vulgaris*

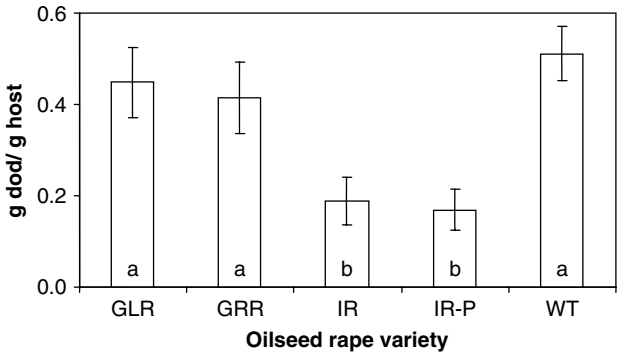

Figure 4. *Cuscuta campestris* development express as the ratio between the g fresh weight of *C. campestris* (dodder) and the g fresh weight of hosts 35 days after sowing the parasite seeds. Each variety had over 40 pots of parasite-infested hosts. WT = wild type; GLR = glufosinate resistant; GRR = glyphosate resistant; IR = imidazolinone resistant (Advanta seed); IR-P = imidazolinone resistant (Pioneer seed). Bars containing the same letter are not different at the $P = 0.05$ level.

reduced the ability of *C. salina* to infest the host.[31] The results of this research suggest that, unlike other parasitic plants,[6,7] *Cuscuta* control is not straightforward, and each parasite–crop system needs to be evaluated on a case-by-case basis to determine the most economical system for managing the parasite, and that, in order to achieve efficient *Cuscuta* control, there should be more emphasis on developing parasite-resistant hosts in addition to herbicide resistance.

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